of the Cammidge reaction. We tried to establish this fact by removing the obstruction in the remaining two dogs of the second series, but were unsuccessful in Dog 9, and in Dog 10 positive reactions were obtained after two negative days. This may have been caused by a closure of the duct, but we have no means of verification, as there were so many adhesions that the pancreatic duct could not be located.

The view that occlusion of the duct might be the main cause of the appearance of Cammidge crystals had to be abandoned after we had completed Series III and IV, as the duct was left patulous in these cases, except in Dog 11 (Series III), and positive reactions were obtained in each case.

Cammidge has asserted that pentose is the specific substance which he isolates from the urine by his method, and that the pentose has its origin in the pancreas. We are not in favor of this view, for, were this correct, we should not expect to find typical crystals after total extirpation of the gland. We are inclined to believe that the substance found by the use of Cammidge's technique is but an expression of disturbed carbohydrate metabolism, this disturbance being referable to morbid changes in the pancreas. We have made no investigations along the lines of chemistry, but the melting point of the crystals found in one case was between 150° and 155° C., which is about the melting point of the phenylhydrazin compound of glycuronic acid.

Conclusions. 1. The Cammidge reaction is a constant feature in hemorrhagic pancreatitis, in mechanical injuries of the gland (crushing of the tail, partial extirpation), and in total extirpation.

2. In certain cases of the subacute type of pancreatitis the reaction is inconstant.

3. The nature of the phenylhydrazin compound is not definitely established. If pentose, it is apparently not derived from the pentose-yielding material of the pancreas.

4. A positive reaction is indicative of altered carbohydrate metabolism due to disturbance of the internal secretion of the pancreas.

THE TONSILS AS ELIMINATIVE ORGANS.

By William W. Ashhurst, M.D.,
of Chihuahua, Mexico.

The true functions of the tonsils and other adenoid structures abutting on the mucous membranes is a problem nearly as obscure today as it was twenty years ago. They were then supposed to secrete a substance which facilitated swallowing the bolus of masticated food, and also to prevent in some obscure way the entrance of infectious materials into the body.

Francis R. Packard[1] writes: "The faucial tonsils are really a large pair of lymphatic glands, the functions of which are but imperfectly understood. As with other lymph glands they probably protect the organism from infections of various kinds by acting as filters, and by in some way lessening the toxicity of microörganisms which come within their sphere of activity."

This seems to represent the sum and substance of the ancient and modern teaching in regard to the matter. But it is somewhat incomprehensible why the microörganisms should enter the tonsils at all, when it is to be supposed that they might just as easily slip by unharmed, into the pharynx and œsophagus. If there were such a thing as ciliated epithelium in the follicles and crypts of the tonsils, one might imagine a continuous current such as exists in the Fallopian tubes, sucking them in against their will. And yet the crpyts of the tonsils even in health are found loaded with a varied flora of pathogenic and non-pathogenic germs—Loeffler's bacilli, pneumococci, streptococci, and staphylococci in pleasing variety. The natural supposition would be, since the nose is constantly exposed to atmospheric infections, and the mouth to those brought by the food and otherwise, that many of these microörganisms failing to penetrate the mucosa of the nose and mouth find lodgement in the tonsillar crypts. This, however, is a very unsatisfactory explanation of their presence there.

The tonsils are composed as we know of adenoid tissues, resembling that of the internal lymphatic glands, as distinguished from the superficial, or those abutting on the mucous membranes, such as the lingual follicles, the tonsils, the solitary and agminated glands of the intestine. The great and evident difference is this, that the superficial lymphatic glands have no direct connection with the lymph vessels, while the deep lymphatic glands are directly in the course of these, and are penetrated by them in all directions. The superficial lymphatic glands are, however, surrounded at their bases by wide lymph spaces, and are described in works of anatomy and physiology as "hanging into" these lymph spaces which communicate by small channels with the larger vessels. That the superficial lymph glands are different in their functions from the deep glands may be inferred from their different arrangement in this respect and from the fact that the deep glands are often found enlarged without any corresponding enlargement of the superficial glands, which may be supposed to be connected indirectly with the same system of channels. The superficial lymph glands have a free blood supply, thus being connected intimately with the blood circulation.

Physiologically there is a certain similarity between the superficial and the deep lymph glands, in that they both have "germinating centres" whence issue enormous numbers of wandering leukocytes. In the deep glands all these leukocytes are poured into the lymph channels whence they are carried into the venous circulation. With the superficial glands the course is different. Some of them are poured into the surrounding lymph spaces, whence they may eventually reach the lymph channels and the venous circulation; but a vast number of them penetrate between the epithelial cells covering the glands, thereby causing an outward current. It is with these escaping leukocytes and the part they play that we are now concerned. Their outpouring into the crypts, and their being found there together with innumerable microörganisms is probably more than a coincidence. It might be regarded as a never-ending succession of sorties by the defenders of the body against the invading horde of microörganisms, and the crypts and surfaces of the glands might be regarded as the battle ground where the infection or protection of the body is decided. But why should the invading microörganisms select such a well-defended part for their onslaught? There lies the whole improbability of the idea.

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[1] Osler's Modern Medicine, 1907, iii, 596.

W. G. McCallum[2] says of the mode of invasion in primary intestinal tuberculosis, "the bacilli adhere to the mucosa, and work their way into its substance. Orth holds that the lesion begins always in the lymph nodules. . . . The first change is of the appearance of tubercle nodules in the substance of the mucosa and very frequently in the lymphoid nodules, or Peyer's patches."

Now so far as we have any positive knowledge, there is no proof that the superficial lymphatic glands ordinarily absorb anything. Of foods, fat is the only one ordinarily absorbed through the lymph channels, but there is not the least evidence that the superficial lymphatic glands have anything to do with this. The fat in the form of soap passes between the epithelial cells to the lacteals, which penetrate the villi, and are nowhere near the follicles and have no connection with them.

Russel H. Chittenten and Lafayette B. Mendel[3] say "there is some evidence that the lymphatic tissue elements are concerned in these synthetic transformations" (re-conversion of soaps into fats), but as the lacteals in the villi are surrounded by adenoid structure unconnected with the lymph follicles, it is the adenoid tissue in the villi that is more likely to be concerned than any other. Fat carrying leukocytes are found in the lacteals in the villi, but so far as I can learn they have never been observed in the lymph spaces surrounding the follicles, nor in other lymph spaces.

So that it seems so far we have no evidence that the superficial adenoid glands absorb anything during the digestive processes. Now if they are only capable of absorbing microörganisms, it would seem that we should be better off without them. But by observing and studying their pathology some new light seems to be thrown on their functions, which will explain many things otherwise difficult to understand, such as McCallum's statement quoted above in regard to the invasion of the intestine by tubercle bacilli, that the tubercle nodules appear "sometimes in the lymphoid nodules or Peyer's patches."

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[2] Ibid., p. 230.

[3] Ibid., I, 695.

Why is it that in diphtheria, scarlet fever, and other exanthemas the tonsils are often affected after other manifestations of the disease have been present for hours or days? This question first forcibly presented itself to me while I was attending a fellow-physician who, in opening a postscarlatinal abscess, received a scratch or prick on the dorsal surface of his right middle finger. The following day he performed an operation on another septic case, and while operating noticed discomfort in the finger. The following day the finger was worse and lymphangitis appeared on the forearm. On the fourth day he called me in. The finger was rather painful and moderately swollen, the swelling extending to the wrist. The red line of the lymphangitis reached the elbow. There was an enlarged and slightly tender anterior axillary gland; and slight fever and general malaise. He had himself freely incised the finger, and was treating it in approved antiseptic style. There was nothing wrong with the throat, nor any symptom of scarlet fever, which he had already had. About two days later the tonsil on the affected side (the right) became inflamed, and presented the typical appearance of scarlatinal tonsil. Two days afterward the left tonsil went through the same process. Convalescence was rather slow, but without notable incident. He himself examined a swabbing from the tonsils with the microscope to see if Loeffler's bacillus was present, but did not find it. Many staphylococci were present. I regret that no bacteriological cultures were made either from the pus from the finger or from the membrane in the throat. When first called I was rather surprised to find nothing apparently wrong in the throat, and was again surprised when the throat trouble appeared. Since the infection was both local and general when I first saw him, it appeared probable to me when the throat trouble developed that this was a form of elimination, like a diarrhœa as the result of an indigestion. The toxins or the toxins and the bacteria together were coming out through the tonsils thereby causing a local manifestation at their point of exit. It seemed that elimination was twofold: local, whereby the nearest tonsil was involved first, and general, whereby the other was involved secondarily. This seemed at first rather hypothetical, but in the light of further thought and observation it seems continually more probable, as explaining points otherwise obscure. Adopting this as a hypothesis, it explains the analogous condition of the solitary follicles and Peyer's patches in typhoid fever, a general and local infection (for the typhoid bacilli are probably absorbed principally from the intestine) with elimination through the nearest superficial lymph glands. It explains the problem of tuberculous nodules in the lymph follicles in primary or secondary tuberculosis of the intestine. It explains the condition of the adenoid structures of the tongue in gastric and œsophageal diseases, of the pharynx in laryngeal affections, and in general the condition of chronic hypertrophy of these organs in all its forms.

It has been observed for years that tonsillar hypertrophy is not simply a local disease, but that the hypertrophy accompanies a diathetic taint or a condition of malnutrition; and that the removal of the hypertrophied organs often greatly benefits the general health is explained in two ways: First, the removal of a mechanical obstruction to respiration and deglutition, and, second, an improvement of the function of the tonsil, whose crypts and recesses have become so deeply situated that the normal outward flow is greatly impeded, and accumulations take place in them, to the detriment of the organ itself and of the whole body.

To some extent the different forms of hypertrophy may be regarded as indicating different forms of overstimulation. The hyperplastic form would indicate excessive elimination of some irritating substance from the general circulation, probably brought to the tonsil by the blood, and the true hypertrophic form would indicate local elimination, probably directly from the lymph space surrounding the tonsils, as in the case of nearby tuberculous disease. To some extent the true hypertrophy must be a compensatory condition, but nevertheless one requiring correction when excessive, as when the lacunæ are so deep that the eliminative function is interfered with.

The obscure relation between rheumatism and follicular tonsillitis will find its explanation in this hypothesis, rather than in the old belief that the rheumatism might enter through the tonsil. Some light ought to be thrown on the causes of appendicitis by this probable function of the adenoid structure in the appendix. Nor shall I be surprised if eventually some explanation along these lines may be found for gastric and duodenal ulcers and various other diseases of the digestive organs whose causes are still entirely obscure.